Original Article

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Role of Blood Ketone Levels in the Diagnosis and Management of Patients with Diabetes Ketoacidosis: A Cross-sectional Study

Internal Medicine Section

GNANASA KALAPALA[1], VIGNESSH RAVEEKUMARAN[2]

ABSTRACT

Introduction: Diabetic Ketoacidosis (DKA), a potentially fatal complication of diabetes, is characterised by accumulation of ketone bodies, metabolic acidosis, and hyperglycaemia. Blood ketone levels serve as key indicators for the diagnosis and management of DKA. The clinical significance of blood ketone cut-off values remains underexplored, necessitating further research to improve patient outcomes.

Aim: To determine the role of blood ketone levels in the diagnosis and management of patients with DKA.

Materials and Methods: The present cross-sectional, hospital-based study was conducted over two years among patients presenting with signs and symptoms of DKA or elevated blood glucose (>250 mg/dL, or ≤250 mg/dL in suspected euglycaemic DKA) were included. Blood ketone levels were measured at baseline, at the time to half-reduction (t1/2), and at resolution, and their diagnostic characteristics were evaluated. Data were analysed using Statistical Package for Social Sciences (SPSS) (v_24.0) software, diagnostic performance of blood ketone measurements was assessed by Receiver Operating Characteristic (ROC) curve analysis.

Results: Among 45 patients, 23 (51.1%) were female. Age distribution was 35.6% (31-50 years) and 31.1% (51-70 years). Urine ketone testing was positive in 53.3% and negative in 46.7%. Mean blood ketone levels were 2.231±1.91 mmol/L at presentation, decreasing to 0.982±1.06 mmol/L after two hours, and 0.253±0.23 mmol/L at resolution. Sensitivity of blood ketone measurement was 100%, specificity 42.9%, Positive Predictive Value (PPV) 66.7%, and Negative Predictive Value (NPV) 100%.

Conclusion: Serum ketone measurement is more reliable than urine dipstick testing for DKA diagnosis. The ketometer is less accurate than conventional clinical assessments and should be used as an adjunctive diagnostic tool in emergencies rather than as a standalone modality.

Keywords: Beta-hydroxybutyrate, Ketometer, Metabolic acidosis

INTRODUCTION

Diabetes Mellitus (DM) is a global pandemic ranked by the World Health Organisation (WHO) as the ninth leading cause of death [1,2]. The burden is particularly high in developing countries such as India, where prevalence is projected to increase from 72 million in 2021 to 134 million by 2045 [3,4]. DM is a metabolic disorder characterised by chronic hyperglycaemia and disturbances in carbohydrate, fat, and protein metabolism due to absolute or relative insulin deficiency and/or resistance [5]. It is classified into Type 1 DM (T1DM), caused by β-cell destruction and absolute insulin deficiency, and Type 2 DM (T2DM), driven by insulin resistance [5,6].

DKA is a hallmark complication of absolute insulin deficiency, more common in T1DM, leading to excessive ketone body production and metabolic acidosis [5,7-9]. Ketone bodies are small, water-soluble lipids produced by the liver, comprising acetoacetate and β-hydroxybutyrate (β-OHB) [10]. According to the American Diabetes Association (ADA), DKA is defined by the triad of hyperglycaemia (blood glucose >200 mg/dL), metabolic acidosis (pH < 7.3 and HCO_3^- < 15 mmol/L), and ketonaemia (β-OHB > 3 mmol/L) [7,11].

Without prompt intervention, intravascular hyperosmolality and cellular dehydration can lead to hypokalaemia, high morbidity, and mortality rates up to 25-30% at T1DM onset and 4-29% in T2DM, with nearly 100% fatality if misdiagnosed or mistreated [7-9,11,12]. DKA is the primary cause of hospitalisation in 16.5-78% of newly diagnosed T1DM patients, especially younger individuals or those without a family history of DM [7,11,12]. Pathophysiologically, insulin deficiency fails to suppress lipolysis, releasing large amounts of Free Fatty Acids (FFA) that undergo hepatic β-oxidation and ketogenesis, resulting in ketosis [13]. Treatment focuses on correcting hypovolaemia, hyperglycaemia, metabolic acidosis, and electrolyte imbalances, with frequent monitoring to restore homeostasis [7,9,11].

Currently, emergency screening for DKA involves urine dipstick testing for ketones, which is inexpensive and rapid but has poor specificity (≈50%), leading to false positives and unnecessary work-ups [14-16]. Nitroprusside-based reagents detect acetoacetate and acetone but not β-OHB [14]. Accordingly, the ADA discourages urine dipstick use and recommends serum β-OHB measurements for DKA screening, as β-OHB is the predominant ketone during decompensation, with the β-OHB: acetoacetate ratio shifting from 1:1 to 3:1 [14-19].

Consequently, the Point-Of-Care Testing (POCT) of ketones measures the concentrations of β-OHB in patients plays a crucial role in the timely identification of DKA. In turn, significantly reduce morbidity and mortality rates while improving patient outcomes [15,17,20]. Nevertheless, POCT testing is only admissible as a screening test and is not recommended for ongoing monitoring of DKA [14,15,17]. Despite advances in ketone monitoring, three key gaps persist where routine urine dipsticks lacks specificity and fails to detect the predominant ketone β-OHB, limiting their utility in DKA screening as explained earlier, then the POCT of capillary β-OHB enables rapid assessment, optimal blood-ketone cut-off values for both diagnosing and monitoring DKA remain poorly defined in real-world settings and the current guidelines restrict the capillary ketone testing to a single screening use and do not address its role in serial monitoring of DKA resolution, leaving uncertainly about the best way to tract the treatment response. With this background, this study was carried out to determine the role of ketone bodies in diagnosis and management of DKA. The primary objective was to

determine the diagnostic accuracy (sensitivity, specificity, PPV, NPV) of various POCT blood ketone cut-off values for DKA diagnosis and the secondary objective was to estimate the proportion of patients with elevated blood ketones who develop DKA within 48 hours and to assess time to DKA resolution using blood gas parameters and ketone levels.

MATERIALS AND METHODS

The present hospital-based analytical cross-sectional study was conducted at the Department of General Medicine, Mahatma Gandhi Medical College and Research Institute, Puducherry, India, from, September 2022 to August 2024. Ethical clearance obtained with Institutional Human Ethics Committee (IHEC) (MGMCRI/Res/01/2021/44/IHEC/82). Informed consents were obtained from all participants, either directly or from a relative if the patient was clinically unstable or unconscious. Re-consent was obtained when the patient's clinical condition improved, or they regained consciousness.

Sample size calculation: Considering the prevalence of DKA among youths with T1DM was 40% [21], for an expected sensitivity of 100% and specificity of 93%, with confidence level at 95% and considering the drop-out rate of 5%, the sample size was calculated to be 45. Using the formula,

$$n = \frac{Z^2_{\alpha/2}\, Sp\,(1-Sp)}{w^2 \times (1-P)} = \frac{1.96^2 \times 0.93\,(1-0.93)}{0.55^2 \times (1-0.04)} = 0.404$$

(Zα/2 - 1.96; expected Sn - 1; expected Sp - 0.93; disease prevalence (p) - 0.04; precision (±expected) - 0.1; w (error around p) - 0.55), calculating with 5% dropout rate it is 42 which was rounded to the nearest highest figure of 45. Consecutive sampling technique was used to include all patients with inclusion criteria until the desired sample size was achieved.

Inclusion and Exclusion criteria Patients above 18 years with DM and the signs and symptoms of DKA or high glycaemic profile (≥250 mg/dL, or <250 mg/dL in suspected euglycaemic DKA) [7], and/or abnormal Arterial Blood Gas (ABG) parameters (pH <7.3, HCO3 <18 mEq/L, Anion gap >10) [7,11] who either presented to the hospital or were already admitted for another purpose were included in the study. Patients with metabolic acidosis due to septicaemia, uraemia {Acute Kidney Injury (AKI) and Chronic Kidney Disease (CKD)} and lactic acidosis due to poisoning were excluded from the study. Thus, a total of 45 patients who met the inclusion criteria were enrolled during screening.

Study Procedure

Data were collected using a semi-structured proforma which included demographic and diabetes details along with relevant clinical data including Glasgow Coma Scale (GCS), dehydration, abdominal tenderness, and laboratory data. At the time of admission, Capillary Blood Glucose (CBG), urine ketones by nitroprusside test, ABG analysis, and capillary β-OHB (by POCT ketometer) were done to diagnose DKA. Those diagnosed with DKA were managed according to institutional protocol with blood ketone levels measured at t1/2 (expected time to reduction of blood ketones to half the baseline value considering a drop of 0.5 mmol/L/hr as an indication of adequate treatment, viz., if blood ketones at admission 6.5 mmol/L, and normal blood ketone value is 0.5 mmol/L, considering an adequate reduction rate of 0.5 mmol/L/hr,

$$Time\ of\ resolution = \frac{Blood\ ketones\ at\ admission - normal\ blood\ ketones}{Normal\ blood\ ketones} = \frac{6.5-0.5}{0.5} =$$

12 *hours*; t1/2 is six hours) and resolution of DKA. Patients diagnosed with DKA had blood ketones measured thrice at baseline, t1/2, and resolution and were followed up with ABG parameters repeated at every six-hour intervals for 48 hours. Patients not diagnosed as DKA were treated as per institutional standard criteria. Confidentiality, privacy, and anonymity of the participants were guaranteed throughout the study.

STATISTICAL ANALYSIS

The collected data were entered in microsoft excel and analysed using Epi-info (ver. 7.2.2.6; centers for disease control and prevention, Atlanta, GA, USA, and World Health Organisation) and IBM Statistical Package for Social Sciences for Windows (SPSS Inc. version 20.0, Chicago, IL, USA). Normality of the variables were measured using Shapiro-Wilk test and Q-Q plot. The data was presented in the form of numbers and percentages for qualitative variables and mean±Standard Deviation (SD)/median with Interquartile Range (IQR) for quantitative variables. Appropriate tests of significance i.e., Chi-square test or Fisher-exact test, Student t-test, Analysis of Variance (ANOVA), or Mann-Whitney test was used to test the significance. Values of $p<0.05$ was statistically significance. Diagnostic test accuracy parameters like Sn, Sp, PPV, NPV, and Likelihood Ratio (LLR) were calculated for POCT blood ketone value. Receiver Operating Characteristics (ROC) curve and Area Under Curve (AUC) analysis were used to determine the best cut-off for diagnosis and resolution of symptoms while on treatment. For all tests, a two-sided p-value ≤0.05 was considered statistically significant.

RESULTS

The sociodemographic characteristics of the study participants were presented in [Table/Fig-1]. DM profile of the study participants was presented in [Table/Fig-2]. The vitals of the study participants shown that the mean heart rate was 104.57±15.8 beats per minute, systolic and diastolic blood pressure was 118.44±15.94 mmHg and 74.22±10.11 mmHg, and the respiratory rate was 20.57±3.72 breaths per minute. [Table/Fig-3] represents the general examination and laboratory investigations of the study participants.

Variables	n (%)
Age (years)	
<30	8 (17.8)
31-50	16 (35.6)
51-70	14 (31.1)
>71	7 (15.6)
Gender	
Male	22 (48.9)
Female	23 (51.1)

[Table/Fig-1]: Socio-demographic characteristics of the study participants (N=45).

Variables	n (%)
Type of diabetes	
T1DM	10 (22.2)
T2DM	35 (77.8)
Clinical symptoms (multiple responses)	
Nausea	21 (46.7)
Vomiting	34 (75.6)
Thirst	13 (28.9)
Polyuria	3 (6.7)
Abdominal pain	22 (48.9)
Shortness of breadth	11 (24.4)
Fever	16 (35.6)
Altered sensorium	3 (6.7)
Similar past history	3 (6.7)
Pregnancy at present	1 (2.2)
Duration of DM (years)	
Newly diagnosed	4 (8.9)
<5	12 (26.7)
6-10	10 (22.2)
>11	19 (42.2)

Family history	
Significant	2 (4.4)
Insignificant	43 (95.6)
Duration of OHA (years)	
<5	18 (40.0)
6–10	8 (17.8)
>11	19 (42.2)
OHA	
Biguanides	32 (71.1)
Sulfonylureas	26 (57.8)
DPP 4 inhibitors	6 (13.3)
SGLT – 2 inhibitors	5 (11.1)
Insulin	
Yes	18 (40.0)
No	27 (60.0)
Duration of insulin (years) (n = 18)	
<5	15 (33.3)
6–10	1 (2.2)
>11	2 (4.4)
History of omission of antidiabetic drug	
Yes	9 (20.0)
No	36 (80.0)

[Table/Fig-2]: Diabetes profile of the study participants (N=45).

Variables	Results
GCS	
11	1 (2.2)
15	44 (97.8)
Dehydration	
Yes	37 (82.2)
No	8 (17.8)
Abdominal tenderness	
Yes	16 (35.6)
No	29 (64.4)
Random Blood Sugar (RBS) (mg/dL)	412.05±103.64
Total WBC count (per microlitre)	13911.11±6618.27
At presentation time	
Arterial pH	7.21±0.11
Arterial pCO_2 (mmHg)	26.95±8.09
Anion gap	22.24±5.62
Electrolytes	
Sodium (mEq/L)	133.38±5.34
Bicarbonate (mEq/L)	11.81±4.64
Potassium (mEq/L)	4.19±0.68
Chloride (mEq/L)	99.51±5.32
At t1/2 /After 2 hours	
Arterial pH	7.34±0.09
Arterial pCO_2 (mmHg)	29.05±6.89
Anion gap	12.04±5.42
Electrolytes	
Sodium (mEq/L)	139.33±9.04
Bicarbonate (mEq/L)	19.81±5.21
Potassium (mEq/L)	4.01±3.68
Chloride (mEq/L)	97.51±5.07
At resolution time	
Arterial pH	7.39±1.01
Arterial pCO_2 (mmHg)	35.95±4.30
Anion gap	10.24±4.22
Electrolytes	
Sodium (mEq/L)	140.91±2.04
Bicarbonate (mEq/L)	22.01±3.21
Potassium (mEq/L)	4.89±1.68
Chloride (mEq/L)	93.11±8.12

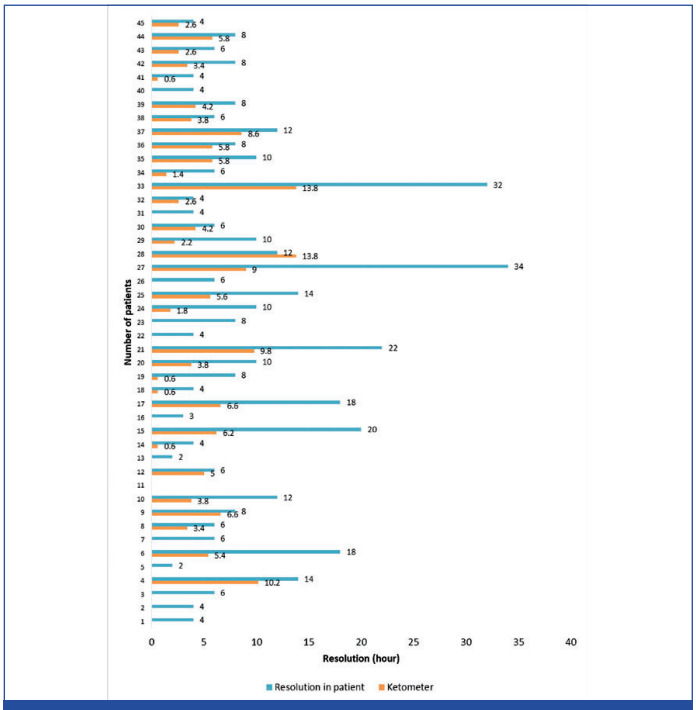
[Table/Fig-3]: General examination and laboratory investigation among the study participants during admission (N = 45).
Categorical variables were presented in number (percentage); while continuous variables were presented in mean ± Standard Deviation (SD); GCS: Glasgow coma scale; pCO2: Partial pressure of carbon dioxide; WBC: White blood cells.

Urine ketone testing revealed that 53.3% of patients were positive, while 46.7% were negative. POCT of blood ketone levels [Table/Fig-4] showed that at presentation, the mean ketone level was 2.231±1.91 mmol/L, which decreased to 0.982±1.06 mmol/L after two hours and further reduced to 0.253±0.23 mmol/L at resolution. The mean resolution time according to the ketometer was 3.56±3.72 hours, with a range from 0 to 13.8 hours. In contrast, the clinical resolution time in patients had a mean of 9.00±7.13 hours (Range 0 to 34 hours). The mean total insulin dose required for DKA resolution was 27.35±20.15 units, with a wide range from 8 to 90 units.

Variables	Mean±SD
Blood ketone levels (mmol/L)	
At presentation	2.231±1.91
At t1/2 /after 2 hours	0.982±1.06
At resolution	0.253±0.23
Resolution hours	
Ketometer	3.56±3.72
Clinical resolution in patient	9.00±7.13
Required insulin dose for DKA resolution (units)	27.35±20.15

[Table/Fig-4]: Blood ketone levels of the study participants (N = 45).
DKA: Diabetic ketoacidosis; SD: Standard deviation

Patients with positive urine ketones had a significantly higher mean blood ketone level at presentation (3.26±1.88 mmol/L) compared to those with negative urine ketones (1.04±1.10 mmol/L), were statistically significant (p <0.001). The mean difference in resolution hours between the blood ketometer and clinical DKA resolution was -5.44±4.91 hours and were significant statistically (p <0.001) [Table/Fig-5].

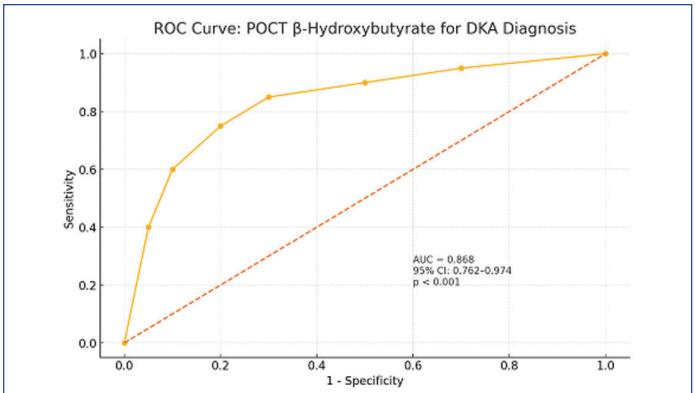


[Table/Fig-5]: Blood ketometer resolution hours vs DKA resolution in patient.

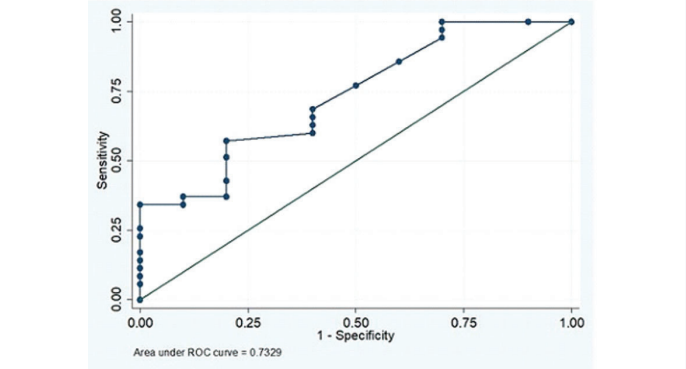
The diagnostic accuracy of the POCT blood ketones resulted that blood ketone measurement is highly effective in identifying true positives (100%) and negatives, although its Sp was low (42.9%), ROC curve analysis established an optimal β-OHB cut-off of 1.5mmol/L for distinguishing DKA from non-DKA states, and presented in [Table/Fig-6,7]. For diagnosing DKA, at the time of admission, the maximum Youden S Index (YI) for the blood ketone value was 0.37 at the cut-off point of 2.2 where the Sn is 57.14% and Sp is 80%, with 62.22% accuracy and in ROC curve, the AUC was 73.29% for the blood ketone values [Table/Fig-8]. Similarly, for the resolution of DKA the maximum YI was 0.48 at the cut-off point of 7.4 with Sn 50% and Sp 97.67%, the accuracy was 96% and the AUC in ROC curve was 49.42%.

Variables	Results
Sensitivity	100%
Specificity	42.9%
Positive Predictive Value (PPV)	66.7%
Negative Predictive Value (NPV)	100%

[Table/Fig-6]: Diagnostic accuracy of the POCT blood ketone (N = 45).



[Table/Fig-7]: ROC curve for POCT blood ketones.



[Table/Fig-8]: The ROC for the blood ketone values at admission for diagnosis of DKA among the study participants.

DISCUSSION

The DKA is a metabolic stress condition that is defined by a pathological imbalance of absolute insulin deficiency and counter-regulatory hormones (glucagon, catecholamines, cortisol, and growth hormone). This results in a triad of marked hyperglycaemia, ketosis, and metabolic acidosis [7,9,11]. These results in altered hormone-sensitive lipase activity, and increased adipocytes. This results in generation of FFA by Triglycerides (TGL) degradation [7,11,22]. In a typical cycle, FFA undergoes β-oxidation to generate acetyl coenzyme A (CoA), later incorporated into the Tricarboxylic Acid (TCA) cycle. Nevertheless, the enzyme systems are overwhelmed by the increased acetyl CoA, absolute insulin deficiency, and FFA breakdown, results in alterations contributes to conversion of acetyl CoA into ketone bodies in the liver. These ketones offer an alternative energy substrate, primarily in the form of β-OHB and acetoacetate, in an approximate ratio of 10:1 [7,9,11,22,23].

In the present study, the majority of the patients, 77.8% had T2DM, whereas only 22.2% of patients had T1DM. The study findings were consistent with the study done by Seth P et al., where 80% of T2DM patients and 20% of T1DM patients were presented with DKA [24]. Another study by Nunes RTL et al., reported that 84.6% patients with pre-existing DM, while only 19.2% subjects reported as newly diagnosed DM was similar to the present study findings [20]. Another study by Bedaso A et al., also found 58.4% patients were T1DM and 41.5% were T2DM patients similar to our study findings [8]. Further, in another study they found the prevalence of DKA among DM patients to be 40% which is close to former studies conducted in Italy (40.3%) [25] and Collared university (38.6%) [26] in T1DM patients. These variations in the incidence levels of DKA might be attributed to the sociocultural differences in the health seeking behaviour, a change in feeding and overall lifestyle emanating from increasing urbanisation and economic development in the region.

DKA presents with various clinical manifestations and sometimes makes it cumbersome to reach a diagnostic outcome when assessed through the symptoms reported by the patient. In The current study, the most common clinical symptoms found were vomiting (75.6%), followed by nausea (46.7%), and abdominal pain (48.9%). Nonetheless, majority of 82.2% of patients were found to be dehydrated and 35.6% presented with abdominal tenderness. Study done by Shahid W et al., reported that nausea and vomiting (57.7%) were the common symptoms followed by pain in abdomen (42.2%) and dehydration (42.2%) which was similar to the current study findings [27]. Another study done by Seth P et al., also reported nausea and vomiting (63.3%) were the common symptoms in DKA were in consistent with the current study findings [24]. Takai et al, also reported that vomiting and abdominal pain (58.3%) were the common symptoms in DKA patients [28]. These shows that in cases of acute ketonaemia, due to insulin omission with increase in the ketone bodies activate the chemoreceptor trigger zone of the medulla oblongata results in vomiting [28,29]. Thus, it is imperative to manage and treat the clinical signs and symptoms associated with DKA to achieve a favourable prognostic outcome and reducing the chance of morbidity.

In the present study, the mean Random Blood Sugar (RBS) was 412.05 mg/dl. Similar results in consistent with the study study findings were reported by Nunes RTL et al., wherein serum glucose was 466 mg/dl [20]. Another study done by Arora S et al., reported the serum glucose levels to be 678 mg/dl which was higher when compared to our study findings [14]. Study by Ahuja W et al., also reported increase in the RBS among the patients with DKA [29]. This shows the pathophysiology of the DKA where the lack of insulin causes elevated glucose levels in the blood by gluconeogenesis process [11,22,30]. This also supports the diagnostic criteria of the DKA.

Similarly, the mean arterial pH of 7.219 and electrolyte levels revealed mean sodium and HCO3 was 133.38 mEq/L and 11.813 mEq/L, respectively and the arterial pCO2 was 26.956 mmHg, and the anion gap was 22.242 in our study. Study by Nunes RTL et al., reported mean arterial pH of 7.18, mean serum sodium and HCO3 was 131.31 and 7.5 mEq/L which was in consistent with This study findings [20]. While study by Arora S et al., reported venous pH was 7.19 and mean serum HCO3 was 8 mEq/l [14]. All these supports the diagnosis of DKA.

We found that patients with positive urine ketones had a significantly higher mean blood ketone level at presentation (3.267 mmol/L) compared to those with negative urine ketones (1.048 mmol/L). Similarly, study done by Taboulet P et al., also concluded that good correlation was observed between the low values of urine and blood ketones, but poor correlation was found on high values similar to our study findings [31]. POCT of blood ketone provide more advantages than urine ketone for the diagnosis of DKA by Kilpatrick ES et al., [32]. This shows that blood ketones provide more reliable findings

than urine ketones by dipstick method.

The mean blood ketone levels were found to be 2.231 mmol/L, which dropped to 0.982 mmol/L after two hours with the endpoint being 0.253 mmol/L at resolution. As the mean resolution time according to the ketometer was 3.56 hours, in contrast to the clinical resolution time in patients who had the mean of nine hours. In terms of resolution of ketone bodies, the mean difference in resolution hours between the blood ketometer and clinical DKA resolution was -5.44 hours, and significant statistically ($p<0.001$). Alwahbi MF et al., found that median time of DKA resolution was 15 hours, which was higher when compared to the present study findings [33].

The diagnostic accuracy plays an important role in determining the accuracy of the disease process; however, a low specificity makes it vulnerable to precisely detect the presence of ketones; it is more reliable than the assessment/detection of urinary ketones [34,35]. In the present study, the diagnostic test accuracy of ketometer showed that at admission and at the time of resolution, the maximum YI for the blood ketone value was 0.37 and 0.48 for the diagnosis of DKA at the cut-off value of 2.2 and 7.4, respectively. The Sn and Sp at the time of admission was 57.14% and 80%, with 62.22% accuracy and at resolution it was 50% and 97.67% with 96% accuracy. AUC was 73.29% and 49.42% in ROC at admission and resolution time. Taboulet P et al., showed that measuring 3β-OHB in capillary blood is faster and more effective than using urine dipsticks for detecting DKA in the emergency setting [36]. Studies by Plüddemann A et al., and Charles RA et al., also supported the use of β-OHB for the detection of DKA in emergency instead of using urine dipstick method [15,17]. While Bektas F et al., found that the Sn and Sp of urine ketone dipstick testing for determining DKA were 66% and 78%, respectively, while for capillary blood ketone testing, they were 72% and 82%, respectively [37]. Tremblay ES et al., also found that β-OHB Sn and Sp was 76.6% and 96.4% indicating its superiority than dipstick methods [19]. This shows that POCT ketone assessment need to be included in the emergency setting with ketometer to assess β-OHB rather than urine dipstick method which used acetate for the diagnosis of DKA.

Even though ketometer is extensively used, it lacks the precise assessments, making it difficult and considered as a non-reliable diagnostic armamentarium. Therefore, the use of ketometer should be more of an adjuvant in case of emergencies, rather than using it as a standalone diagnostic modality like the conventional clinical parameters.

Considering the management of DKA, mostly managed with an intravenous (i.v.) insulin infusion until resolution, and once the patients start to eat and drink normally, i.v. will be shifted to subcutaneous insulin [7,11,22,30]. In the present study, the mean total insulin dose required for DKA resolution was 27.356 units (range 8 to 90 units). Bedaso A et al., in their study reported that about 75.6% of patients were managed by taking short acting agents, followed by the use of intermediate acting agents in 24.3% DKA patients [8]. A pooled study by Alshurtan KS et al., stated that insulin infusion reduces the resolution time of acidosis significantly in DKA patients [38]. Joint British Diabetes Societies for Inpatient Care guidelines recommend that if the concentration of glucose drops below 14 mmol/L, then the infusion rate to be de-escalated to 0.1 to 0.05 units/kg/hr [39].

Limitation(s)

The limitation of the study includes that this study was done at a single centre with a relatively small number of patients, hence the validity of the study at its present form might not be useful to the general population. Secondly, even though we assessed most of the parameters in the subjects, we could not precisely establish an exact cut-off value due to the presence of many confounding factors and lack of a consensus between our study results and the results obtained/reported by various researchers. Thus, we recommend a well-planned multi-centric study with randomised sampling and assessment with a standard operating procedure and observational and reporting guidelines, to set a standard cut-off value.

CONCLUSION(S)

In this study, POCT serum β-OHB measurement achieved 100% sensitivity and NPV in detecting DKA, markedly outperforming standard urine dipstick testing. ROC curve analysis established an optimal β-OHB cut-off of 1.5mmol/L for distinguishing DKA from non-DKA states, while serial monitoring of β-OHB levels closely mirrored biochemical resolution and provided a reliable marker for tailoring treatment. Integrating blood ketone assays into diagnostic protocols can expedite DKA diagnosis and reduce unnecessary work-ups, although these assays should complement rather than replace comprehensive clinical assessment and ABG. Ultimately, implementing POC ketone testing in emergency settings may enhance patient outcomes by enabling more timely and accurate management.

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Authors contribution: Conceptualisation, VR; Methodology, VR; Software, GK; Validation, VR; Formal Analysis, GK; Investigation, GK; Resources, GK.; Data Curation, VR; Writing – Original Draft Preparation, GK; Writing – Review & Editing, VR; Visualisation, VR; Supervision and Project Administration, VR.

Use of AI and AI-assisted technologies in the writing process: The writing process did not involve the use of AI or AI-assisted technologies.

Data Availability Statement: All data generated or analysed in this study are included in this published article.

Ethical approval: The authors are responsible for all facets of the work, ensuring that inquiries regarding the accuracy or integrity of any component are thoroughly investigated and addressed. All procedures conducted in this study adhered to the ethical standards set forth by the relevant institutional and/or national research committees and complied with the Helsinki Declaration (revised in 2013). Informed consent was obtained from the patient for the publication of this case report and associated images. The Institutional Ethical Committee approved this study.

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PARTICULARS OF CONTRIBUTORS:
1. Resident, Department of General Medicine, Mahatma Gandhi Medical College and Research Institute, Sri Balaji Vidyapeeth (Deemed to be University), Puducherry India.
2. Assistant Professor, Department of General Medicine, Mahatma Gandhi Medical College and Research Institute, Sri Balaji Vidyapeeth (Deemed to be University), Puducherry India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Vignessh Raveekumaran,
Assistant Professor, Department of General Medicine, Mahatma Gandhi Medical College and Research Institute, Sri Balaji Vidyapeeth (Deemed to be University), Pondicherry, India.
E-mail: vignesshravee@gmail.com

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